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KIRKPATRICK & LOCKHART NICHOLSON GRAHAM LLP/WYETH STATE STREET FINANCIAL CENTER ONE LINCOLN STREET BOSTON, MA 02111-2950			SALMON, KATHERINE D	
			ART UNIT	PAPER NUMBER
			1634	

DATE MAILED: 09/08/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)
	10/686,619	O'TOOLE ET AL.
	Examiner	Art Unit
	Katherine Salmon	1634

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) Responsive to communication(s) filed on 16 June 2006.
- 2a) This action is FINAL. 2b) This action is non-final.
- 3) Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) Claim(s) 1-8 and 22 is/are pending in the application.
- 4a) Of the above claim(s) 4 is/are withdrawn from consideration.
- 5) Claim(s) _____ is/are allowed.
- 6) Claim(s) 1-3,5-8 and 22 is/are rejected.
- 7) Claim(s) _____ is/are objected to.
- 8) Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) The specification is objected to by the Examiner.
- 10) The drawing(s) filed on _____ is/are: a) accepted or b) objected to by the Examiner. Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a). Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) All b) Some * c) None of:
 1. Certified copies of the priority documents have been received.
 2. Certified copies of the priority documents have been received in Application No. _____.
 3. Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) Notice of References Cited (PTO-892)
- 2) Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date 2/6/05, 7/8/05.
- 4) Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____.
- 5) Notice of Informal Patent Application (PTO-152)
- 6) Other: _____.

DETAILED ACTION

1. This action is in response to the papers filed 6/16/2006. Currently Claims 1-8 and 22 are pending. Claims 9-21 have been canceled. Claim 4 has been withdrawn. Claims 1-3, 5-8, and 22 are currently under examination.
2. The following rejections are either newly applied as necessitated by amendment or are reiterated. Response to arguments follows.
3. This action is FINAL.

Withdrawn Objections

4. The objection to the Claims with regard to the misspellings in Claim 2 of the previous office action is moot in view of the amended claims removing the misspelling.

Withdrawn Rejections

5. The rejection of Claims 1,2, 5, 6, and 8 under 35 USC 102(b) made over Mishima et al. in the previous office action, is moot in view of the amended claims. In particular Claim 1 has been amended to diagnosing lupus.
6. The rejection of Claims 1-3 and 5-8 under 35 USC 103(a) made over Swiniarski et al. in view of Takada et al. in further view of Affymetrix GeneChip Murine 11K set is moot in view of the amendment to the claims. In particular, Claim 1 has been amended to detecting an elevated expression level.

New Grounds of Rejection Necessitated by Amendment or Reiterated

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

7. Claim 3 is rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

With regard to Claim 3, it is unclear how the claim is further limiting. Further Claim 1, defines the sample as having LN, it is unclear if the sample has LN how to limit the sample only to SLE.

Claim Rejections - 35 USC § 112-Scope of Enablement

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 1-3, 5-8, and 22 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of diagnosing lupus nephritis (LN) in a mouse wherein the method comprises: 1) obtaining a kidney sample

from a control mouse and a mouse with LN; 2) determining the mRNA transcript level of midkine, 3) comparing mRNA transcript level of midkine between a control and mouse with LN, wherein an increase in midkine mRNA transcript level, relative to the control, indicates that said mouse has an increased likelihood of LN does not reasonably provide enablement for methods to diagnose systemic lupus erythematosus (SLE) or LN in mouse or human by detecting an elevated expression level of midkine gene. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The following factors have been considered in formulating this rejection (*In re Wands*, 858F.2d 731, 8 USPQ2d 1400 (Fed. Cir. 1988): the breadth of the claims, the nature of the invention, the state of the prior art, the relative skill of those in the art, the predictability or unpredictability of the art, the amount of direction or guidance presented, the presence or absence of working examples of the invention and the quantity of experimentation necessary.

Breadth of the Claims

The claims are broadly drawn to diagnosing lupus nephritis in a human or a mouse comprising detecting the expression level of midkine gene in a kidney sample, urine sample, and blood sample wherein an elevated expression level indicates an increased likelihood of lupus nephritis. The claims are broadly drawn to both human and mouse, in three biological samples, and with an undefined elevated expression

level. Further, the “control mammal” of Claim 2 is broadly drawn to ANY mammal. Claim 6 is drawn to ANY tissue sample.

The invention is in a class of inventions that the CAFC has characterized as “the unpredictable arts such as chemistry and biology” (Mycolgen Plant Sci., Inc. v Monsanto Co., 243 F.3d 1316, 1330 (Federal Circuit 2001)).

Teachings in the Specification

The specification teaches systematic lupus erythematosus (SLE) is an autoimmune disease that has diverse and variable clinical manifestations that range from skin rash and joint pain that can show spontaneous remissions to severe kidney disease that may result in renal failure, otherwise known as lupus nephritis (LN). Midkine (MDK) has several functions including neural-glial interactions in brain development, inflammation, tumor and angiogenesis, and anti-apoptotic activities (specification, pages 14-19). The specification asserts that midkine is a marker for SLE or LN, and its expression can be utilized as a diagnostic for said diseases (page 4). The specification concludes “MDK has not previously been associated with SLE and LN....While mouse models were used for the initial differentiation expression analysis, it is well-appreciated that animal models can be interpreted to reflect expression levels from human subjects as well. The present invention...encompasses human MDK” (page 22). The specification further asserts “without limitation as to mechanism, the present invention is based in part on the principle that modulation of the expression of

the MDK gene expression may ameliorate SLE/LN, when they are expressed at levels similar or substantially similar to normal non-diseased tissues" (page 23).

The specification discloses working examples of the isolation of RNA from kidney samples of several different mouse models of lupus that ranged in age of five months to 8, 16, 20 weeks of age, thus representing early, intermediate, and late stages of lupus, and control mice of the same age range. The working examples disclose that after the isolation of kidney tissues from said mice, RNA was isolated and cDNA was synthesized, and then the samples were analyzed with Affymetrix Mu11KsubA and Mu11KsubB microarrays. Statistical analysis was subsequently performed, and TaqMan assays were performed on genes of interest (pages 13-14 and 78-82).

State of the Prior Art

The art acknowledges midkine is a ubiquitous gene that plays an integral component in many cellular pathways and it is expressed in several tissues. Midkine is a member of heparin-binding growth/differential factor family and plays a role during development (Zhang, Current Opinion in Hematology, 1999, Vol. 6, No. 1, page 44 [pages 1-13 in printed HTML article]). Zhang teaches midkine is expressed during embryogenesis in neural development and it is mitogenic in some cell lines (page 7, and Takada et al., Journal of Biochemistry, 1997, Vol. 122, pages 453-458). In adults, midkine plays a role in neural repair and regeneration as it is expressed in response to neural injury, Alzheimer's plaques, and prevents retinal degeneration due to prolonged sun exposure (Zhang, page 7). Sato et al (Journal of Immunology, 2001, Vol. 167, pages 3463-3469) teaches midkine enhances migration of inflammatory cells in

response to renal injuries. Despite its varied role, normal midkine expression in humans is tissue specific. Tsutsui et al teaches (Cancer Research, 1993, Vol. 53, pages 1281-1285) "in normal tissues, MK is weakly expressed in the kidney...lung alveoli...mucosal tissues of the stomach... colon..., and spleen, ...moderately in the thyroid...and highly in the mucosal tissue of the small intestine....No MK mRNA was detected in the liver" (page 1282, Figure 1).

Lupus is an autoimmune disease that is characterized by joint pain, rash, weakness, and primarily affects women (Kotzin, Cell, 1996, Vol. 85, pages 303-306). The underlying cause of lupus has yet to be determined as environmental factors such as sun exposure, viral or bacterial infections, hormonal and drug treatments, and genetic contributions play a role in the manifestation of the disease (Kotzin, page 305). Kotzin teaches several animal models have been used to study lupus, however, due to the complex nature of the disease, "even when one animal model and one phenotype is considered, the genetic basis of lupus-like disease is remarkably complex, involving contributions from multiple genes in addition to class II MHC....Furthermore, it seems likely that different genetic contributions are operative in different animal models (and therefore in different patients), even when the same phenotype is being followed" (page 305). Kotzin further teaches mouse models are used to study the genetic causes of lupus, and to predict human genes that are associated with said disease since mouse and human genes are homologous (Journal of Clinical Investigation, 1997, Vol.99, No. 4, pages 557-558). However, as stated above, environmental factors and phenotypic expression of lupus have considerable variation, and since the environment conditions

are controlled for animal studies and the animal models are bred to have uniform lupus symptoms, it is unclear if results from animal studies can be applicable to humans. Kotzin teaches, “disease phenotype among mice in each cross is much more uniform compared to the relatively heterogeneous disease expression in patients. Especially in SLE, clinical manifestations and autoantibody production can be extremely diverse and variable, which is in part genetically based, and this variability can confound genetic studies” (Journal of Clinical Investigation, page 557). To ensure accurate predictions of the results of mouse lupus models to humans “there should also be concern that an initial mapping in a complex trait reflects false positive readings....If true, this human locus...may not be in a region synthenic to the murine susceptibility locus, and linkage in the current human study would therefore represent quite a fortuitous finding,” and in order to ensure accurate results, large studies of human patients will need to be performed (Kotzin, Journal of Clinical Investigation, page 558).

The Relative Skill of Those in the Art

The level of skill in the art is deemed to be high.

The Predictability or Unpredictability of the Art and Degree of Experimentation

The art is extremely unpredictable with regard to midkine’s expression levels in any biological sample of any mammal. Tsutsui teaches that normal levels of midkine are different among human tissues. With non-uniform levels of midkine in tissues, it is unpredictable how midkine’s expression level relative to controls of specific tissues can be extrapolated to midkine levels of different tissues to detect lupus. Further, the specification is silent about the range or level of wildtype or variant midkine expression

is needed to diagnose SLE or LN. While the skilled artisan can individually determine tissue or sample specific midkine's expression by its presence, absence, upregulation or downregulation in natural state, and compare it midkine's presence, absence, upregulation or downregulation in the same and/or different samples of a diseased mammal, and quantitative the threshold or midkine's range level that is necessary to diagnose lupus such further research is unpredictable and undue. For instance, as Tsutsui teaches above, midkine is not expressed in liver tissues, and it is moderately expressed in the thyroid. It is unpredictable if the upregulation of midkine in the liver can predict lupus if there is no midkine expression in thyroid samples, and if in said example, the levels can be extrapolated to different tissues, it is unpredictable if a 2-fold, or 10-fold level difference can predict lupus. The specification has taught elevated levels of midkine expression in kidney samples can predict LN in mice; however, it is silent about the midkine levels in the remaining biological samples in other mammals that can predict SLE or LN. Moreover, as indicated by Kotzin, an animal model may not be an accurate representation of another animal's response to lupus. Genetic homology does not necessarily correlate to phenotypic expression. As mentioned previously, environmental factors play a role in the development of lupus, and it is unpredictable if a mouse, particularly in a controlled environment, will react in the same manner to environmental factors as humans. Consequently, it is unpredictable if a mouse phenotypic expression of lupus will be similar to humans. Consequently, the skilled artisan would have to examine midkine's expression in any biological samples of any mammal in order to diagnose lupus. Due to the absence of information regarding

other mammalian levels of midkine in various samples, it is highly unpredictable if the presence and absence of midkine and its range of upregulation or downregulation can predict SLE or LN as each mammal and biological sample with the exception of increased midkine expression levels in mouse kidney samples of LN, would have to be individually examined. In view of the quantity of experimentation of detecting midkine expression in any biological sample in any mammal, and determining if its presence or absence, upregulation or downregulation of wildtype or variant form is a lupus marker as well as unknown genetic and environmental variables that cause lupus, reliability mouse models, it is unpredictable if midkine expression levels can be used to diagnose lupus in any biological sample of any mammal. As a result, the specification does not teach the person skilled in the art how to reasonably predict, without undue burden, SLE or LN by midkine expression levels in biological sample of any mammal.

Amount of Direction or Guidance Provided by the Specification

There are no sufficient teachings or quantitative data that describe possible combinations of mRNA levels of midkine expression, its presence or absence, and range of upregulation and downregulation of its wildtype or variant form, in any biological samples of any mammal that can detect SLE or LN other than elevated mRNA transcript levels of midkine in a mouse with LN in kidney samples. The specification does not teach any threshold level of midkine expression is needed to predict lupus, and if the value is species specific and/or specific to biological samples. Further, the specification does

not teach if mutant midkines can also diagnose lupus, consequently, the skilled artisan would not know which specific mutant, a splice variant or single nucleotide polymorphism, for example, and its exact mutation, is needed to prognose lupus. As a result, the skilled artisan would not know if the absence, presence, upregulation or downregulation of wildtype or mutant midkine is necessary to predict lupus and at which stage. The skilled artisan can individually examine any tissue, blood, or urine sample in any mammal such as monkeys, sheep, dogs, pandas or any bodily sample such as cerebrospinal fluid, urine, saliva, and tissues such as brain, stomach, lung, and liver to detect midkine expression and its relationship to lupus, the outcome of such research cannot be predicted. The specification does not teach the person skilled in the art how to reasonably predict, without undue burden, methods of detecting LN with the exception of elevated midkine levels in mouse kidney samples.

Working Example

The specification does not provide working examples of methods to diagnose lupus with midkine expression levels in any biological sample of any mammal afflicted with lupus except for elevated levels of midkine in mouse kidneys. The methods do not demonstrate the methodology can be used to predictably diagnose lupus with any midkine transcript levels of any biological sample in any mammal except for mouse kidney samples.

Conclusions

Case law has established that '(t)o be enabling, the specification of a patent must teach those skilled in the art how to make and use the full scope of the claimed invention without 'undue experimentation.'" In re Wright 990 F.2d 1557, 1561. In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970) it was determined that '(t)he scope of the claims must bear a reasonable correlation to the scope of enablement provided by the specification to persons of ordinary skill in the art". The amount of guidance needed to enable the invention is related to the amount of knowledge in the art as well as the predictability in the art. Furthermore, the Court in Genentech Inc. v Novo Nordisk 42 USPQ2d 1001 held that '(l)t is the specification, not the knowledge of one skilled in the art that must supply the novel aspects of the invention in order to constitute adequate enablement". In view of the high level of unpredictability in the art and lack of guidance provided by the specification and prior art, undue experimentation would be required to practice the claimed invention.

Response to Arguments

The response traverses the rejection. (A) The response asserts the amended claims recite that the mammal is from the group consisting of a human and a mouse. The response asserts the claims "bear at least a reasonable correlation to the scope of the enablement" (p. 6 3rd full paragraph). The response asserts that it is well within the level of skill in the art to apply methods and knowledge from animal studies to humans, despite the differences between controlled animal studies and observed human

diseases (p. 7 last paragraph). (B) The response asserts the claims have been amended to recite a biological sample selected from the group consisting of kidney, urine, and blood (p. 8 1st paragraph). The response asserts that urine and blood are fluids that contact the kidney and have been demonstrated to carry other protein and nucleic acid markers of other diseases (p. 8 1st paragraph). The response asserts that the inventors did foresee and describe implementing their invention by detecting of an expression level of midkine gene in a kidney, urine, or blood sample isolated from a human (p. 8 last paragraph). The response asserts the claims bear "at least a reasonable correlation to the scope of enablement provided by the specification" (p. 8 last paragraph and p. 9 1st sentence). These arguments have been thoroughly reviewed but are not found persuasive.

(A) To be enabled for a method of diagnosing lupus nephritis in a human, the specification does not have to reduce to practice but must describe the method in a way that the skilled artisan could make and use the method without undue experimentation. The specification asserts correlation of expression of a mouse midkine gene with lupus. The specification asserts that the mouse gene and the human gene have "similar" homology. However, in order to make a correlation between a human gene and a disease undue experimentation by the skilled artisan must be performed. The skilled artisan would need to determine if any variant of any fragment of the midkine gene would have the same level of expression in both mouse and human so as to be diagnostic for lupus. The skilled artisan would need to determine the necessary amino acids needed to define the midkine gene in both animals and determine if changes in

the amino acids would have the same change in functionality in both animals. Further, the skilled artisan would also have to determine if population stratification would affect expression levels of the midkine gene in human.

The reply points to Kotzin et al “the results from animal models could be useful in...major ways” including testing the identified genes from linkage association with human disease (p. 7 of reply). The association of human to an expression level of a gene determined by a mouse model requires undue experimentation by the skilled artisan as evidenced by Kotzin et al. Kotzin et al. teaches that lupus is a complex trait resulting from multiple contributing genes (p. 557 1st column 2nd full paragraph). Kotzin et al. teaches there are many factors, which complicate the genetic analysis of lupus (p. 557 1st column 2nd paragraph). Kotzin et al. teaches that different combinations of genes, whether in different ethnic groups or even in the same family may result in the identical disease phenotype with genetic heterogeneity (p. 557 1st column 2nd paragraph). Kotzin et al. teaches that animal models provide an opportunity to control environmental exposures so that disease is solely a reflection of the genes inherited (p. 557 2nd column 1st paragraph last sentence). Therefore Kotzin et al. teaches that animal models reflect ONLY genetic inheritance. It is unpredictable what effect the environmental factors will have on expression of the midkine gene. Further, Kotzin et al. teaches that initial mapping in a complex trait reflects false positive findings (p. 558 1st column last paragraph 1st few sentences). Kotzin et al. teaches even if the mapping indicates a relationship between the murine gene and the human gene, the human gene may not be in a region syntenic to the murine locus (p. 558 1st column last paragraph 1st

few sentences). Expression of genes is affected by surrounding genes. It is unclear if the expression of midkine gene would be same in mouse and human because it is not clear from the specification that the midkine gene is in a similar region in a human as a mouse.

The claims are not all limited to mouse and human. The control mammal in claim 2 can be broadly drawn to any animal. It is unpredictable that ANY mammal could be used as a control to determine elevated expression level in mouse or human.

(B) The argument asserts that because blood and urine is processed by the kidney any expression levels of genes would be the same in kidney, urine, and blood. This is unpredictable because the specification has not taught that expression in the three sample types is similar. Furthermore, if it was the case that because blood is processed by the kidney expression levels are similar, then would any tissue that uses the blood processed by the kidney have the same expression levels? The group of kidney, blood, and urine samples is not a "reasonable correlation to the scope" because the specification and the art do not indicate that expression levels between the three are similar. The reply asserts that urine and blood have demonstrated to carry other protein and nucleic acid markers of other disease and any experimentation to investigate midkine levels in blood and urine would be reasonable and routine. Though, markers of other disease are carried in blood and urine it has not been shown that midkine gene is expressed in blood and urine. Further, it is undue experimentation to determine if the expression of midkine gene is increased in blood and urine and if this increase is due to

environmental or other genetic effects or by lupus. The skilled artisan would have to determine the threshold for the expression of the midkine gene in both blood and urine and determine that would diagnosis a subject as having lupus. It is unpredictable that the same expression threshold could be used by a kidney sample and blood or urine because each sample has different genetic influences on expression.

The claims are not all limited to mouse and human. The samples in claim 6 can be broadly drawn to any tissue sample. It is unpredictable that ANY tissue sample would have the same elevated expression.

(C) As indicated above the scope of enablement is drawn to a comparison of the transcript level of midkine between a control and a mouse. Claim 1 is drawn to detecting an elevated expression level without comparing it to any control. It is, therefore, still unclear as to the limitations of elevated expression level. Detecting an "elevated expression level" can be broadly interpreted as detecting any expression level because the claims have not provided a control in which to compare the expression of midkine. It is unpredictable that ANY elevated expression level will diagnosis lupus.

Conclusion

9. Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

10. A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Katherine Salmon whose telephone number is (571) 272-3316. The examiner can normally be reached on Monday-Friday 8AM-430PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.



Katherine Salmon
Examiner
Art Unit 1634



BJ FORMAN, PH.D.
PRIMARY EXAMINER